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In the Specification

Please delete the paragraph at page 5, lines 3 through 4.

Please replace the paragraph at page 4, line 23 through page 5, line 2 with the following paragraph:

Figure 5A illustrates the P1 peptide amino acid sequence, aligned over a diagram of the hsp65-P1 fusion protein (P1 is shown at the C-terminus of hsp65). When liberated from P1, SIYRYYGL (SEQ ID NO: 1) (demarcated by arrows) binds to K^b to form the peptide-MHC complex recognized by the 2C TCR. In P1, SIYRYYGL (SEQ ID NO: 1) is flanked 5' and 3' by sequences that lie immediately upstream and downstream, respectively, of peptide bonds that are cleaved (see arrows) in murine cells to liberate naturally occurring peptides (SIINFELK (SEQ ID NO: 2) from ovalbumin (Ova) and LSPFPFDL (SEQ ID NO: 3) from α -ketoglutaraldehyde dehydrogenase (α KG) (Falk, K., *et al.*, *Eur. J. Immunol.*, 22:1323-1326 (1992); Ukada, K., *et al.*, *J. Immunol.*, 157:670-678 (1996))).

Please replace the paragraph at page 5, lines 5 through 12 with the following paragraph:

Figure 5B is a pair of histograms, which display experimental evidence that P1 and hsp65-P1 are processed intracellularly to yield the SYRGL (SEQ ID NO: 4) octapeptide. 48 hr after transfection with mammalian expression vectors (VR1055 and pCINeo), containing sequences that encode P1 and hsp65-P1, respectively, EL4 cells were incubated for 18 hr with an equal number of naive 2C T cells. Histograms show the percentage of live, 2C⁺CD8⁺ cells that were stimulated to upregulate the activation marker CD69. The responses of these naive T cells to control EL4 cells, transfected with the empty (vector) plasmids, are shown as shaded histograms.

Please replace the paragraph at page 5, lines 13 through 17 with the following paragraph:

Figure 5C is a graph, which displays experimental evidence that normal C57BL/6 mice have T cells that can recognize the SYRGL-K^b complex. A CD8⁺ T cell line, derived from C57BL/6 mice immunized with the SYRGL (SEQ ID NO: 4) peptide in adjuvant, specifically lysed T2-K^b target cells in a peptide-dependent manner. A highly cytolytic long-term cultured 2C CTL clone (L3.100) is shown for comparison.

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Please replace the paragraph at page 5, lines 18 through 21 with the following paragraph:

Figure 6A is a graph showing CD8⁺ CTL that recognize the SYRGL-K^b complex are produced in C57BL/6 mice injected with hsp65-P1 in PBS but not in those injected similarly with equimolar amounts of various controls (a mixture of P1 and hsp65, the SYRGL (SEQ ID NO: 4) octapeptide, the P1 polypeptide itself, or hsp65 itself; as noted further below, SYRGL is referred to as an "octapeptide" as it is an abbreviation of the sequence SIYRYYYGL (SEQ ID NO:1)).

Please replace the paragraph at page 5, lines 22 through 26 with the following paragraph:

Figure 6B is a graph illustrating the production of SYRGL-specific CTL in mice injected with [a] various amounts of hsp65-P1, 0.015-1.5 nmoles (1-100 µg) or a control fusion protein in which P1 is linked to the C-terminus of a maltose-binding protein (Mal-P1, 80 µg); lysis of T2-K^b target cells in the absence of added SYRGL (SEQ ID NO: 4) peptide is indicated by unfilled symbols.

Please replace the paragraph at page 7, lines 10 through 15 with the following paragraph:

Figure 9B is a pair of graphs illustrating the behavior of hsp65 fusion protein-activated dendritic cells in vivo. Myeloid dendritic cells from lymph nodes draining a subcutaneous site where hsp65-P1 was injected 24 hr previously show increased expression of MHC-1 (K^b) (lower panel) compared to myeloid dendritic cells from lymph nodes draining an uninjected site ("no treatment", upper panel).

Amendments to the specification are indicated in the attached "Marked Up Version of Amendments" (pages i - ii).